

PHILADELPHIA NEUROLOGICAL SOCIETY.

A stated meeting of the Society was held Monday evening, April 28th, the President, Dr. S. WEIR MITCHELL, in the chair.

Dr. J. HENDRIE LLOYD read a paper entitled "The Report of a Case of Pseudo-Hypertrophic Muscular Paralysis With Bone Lesions."

Sarah C., æt. seventeen, was one of the first inmates of the Home for Crippled Children, after its establishment by Mrs. Innes, in the early winter of 1883. The patient presented, when first seen, an appearance of great obesity. The fat was generally distributed to the face, neck, trunk, gluteal regions, arms (partial) and legs. Some of the muscles of the forearms were slightly wasted. The appearance was one of great robustness of health, and the accumulation of fat and general outlines of the figure were such as to suggest an over-fed, middle-aged woman, rather than a girl but half advanced in her teens. Upon closer inspection there was discoverable a strange contrast to this appearance of health. It could be readily seen, then, that the patient was a complete cripple. So absolute was the loss of power that she either lay supine or was propped with pillows in a large chair, the body spreading out, as it were, by the mere force of gravitation, and without muscular resiliency or the control of will-power.

The girl was, and now is, so helpless that it is impossible for her to assume the sitting from the reclining position ; she cannot turn upon her side without assistance, and it is with the greatest difficulty that as she lies upon her back she crosses one leg over the other. She retains, however, some power in her arms, especially the forearms, and although these regions are evidently wasting, they alone seem to possess what fraction of power the girl can command, and with them she can sew, crochet, and do other work requiring skill and training.

The details of her condition are briefly as follows :

The muscular system presents in many places marked enlargements. These are evident especially in the deltoid, lumbar muscles, and calf. The muscles in these regions have a dense, doughy feel, and are overlain with subcutaneous fat, with which they appear so intimately connected that it is difficult to detect their exact limitations. They are very little, if at all, subject to the will, so that their passive condition and their envelope of mottled and lifeless-looking skin are very suggestive, to one who handles them, of some profound pathological change.

This pathological change has been demonstrated by the microscope, and is in accord with the classical description of pseudohypertrophic muscular paralysis. With the aid of the little instrument of Dr. Harte, of this city, specimens of the muscular tissue were taken from the lumbar region and the calf. Dr. William E. Hughes kindly prepared these for examination, by teasing them, as the fragments were too small for embedding and cutting. The specimen from the calf presented under the lens an interstitial hyperplasia, with granular changes in the muscular fibre, and a marked loss of striation. There was not any appearance of secondary fatty change in the connective neoplasm, which may possibly be accounted for by the "teasing" process. The specimen from the lumbar region was not apparently from the muscle, but was simply adipose tissue.

The reactions to the faradic current in the deltoid and calf muscles are very feeble. The reactions with sponges on median and ulnar nerves of forearm—the muscles of which are wasting—are about normal, or, at least, much more active than in the hypertrophied muscles. So, too, in the peronei. The reactions of degeneration to the galvanic current have not as yet been sought for.

The girl was weighed some months after her admission to the Home, and after she had evidently lost much flesh, and turned the scale at 124 lbs. When first admitted it required the combined strength of two men to carry her up stairs, and she, no doubt, at that time weighed considerably more. Her stature is not tall, and her age at that time not seventeen. The measurements of her limbs are at present as follows :

UPPER EXTREMITIES.

Arm, right, $12\frac{1}{2}$; forearm, 9

Arm, left, 13 ; forearm, 9

LOWER EXTREMITIES.

Right thigh, 21 ; leg, $13\frac{1}{2}$

Left thigh, $21\frac{1}{2}$; leg, 13

These figures show an increase of bulk during the last few months. The girl's mind is fairly bright. She is something of a giggler—simpering and foolish,—but her defects may be partly, at least, accounted for by lack of opportunity and education. She cannot be called a feeble-minded child. The patient's history is very obscure. Her mother "ran away with another man," as her brother explains in a letter, and she has passed her life in public institutions. This brother says he thinks she was always a cripple, but not so bad but that she could at one time walk. Her general health is excellent; she attempts to add constantly to her exuberant proportions with a vigorous appetite, and is now putting on fat. The heart and lungs are normal. She occasionally suffers pain from the spinal curvature, to which reference will be made in a moment. There is no evidence of heredity. She at one time menstruated very freely, and as often as every two weeks.

But while the appearance of the muscles is so significant, there are changes in the bones of still greater interest, and to which I desire to call your attention. These changes have occurred in the spine, and in the elbow- and knee-joints. The spine is in a state of constant scoliosis, with its greater convexity to the right. One year ago, this convexity could be thrown to the left by one attendant lifting the patient by the shoulders, and another making pressure on the vertebræ. It would not remain thus, however, and was evidently uncomfortable to the girl.

The elbow-joint presents an unique appearance, which, as far as I am at present informed, has not been described in any other reported case of pseudo-hypertrophic muscular paralysis. It is exceedingly mobile, due apparently to an atrophy of the epiphyses of the bones. Thus, the joint can be bent backward at a marked angle, quite impossible when the olecranon and its opposing surface are intact. The attachments of the head of the radius and upper end of ulna are loosened, allowing a sort of wobbling and limberness in making pronation and supination. This preternatural mobility is increased by a somewhat similar condition of the head of the humerus in the glenoid cavity.

The wrist-joint, also, is not firm and staunch (depicted by Dr. Taylor). The chief defect of the knee-joints is an atrophy of the patellæ, which are not more than one half their normal diameters.

In bringing this case to your notice, I desire especially to call your attention to the fatty phenomena and to the bone lesions. The literature of the subject presents nothing

which has added much to the original description of Duchenne, supplemented by a few very thorough post-mortems—especially of Eulenberg and Cohnheim. Duchenne said that the interstitial connective tissue proliferated with the production of fibrous tissue, and that this increase was associated with fat-vesicles; also that the striation was preserved, but was very faint. He does not bring into prominence the fatty changes, which seem to be secondary to the hypertrophy of the connective tissue. Other writers, apparently following Duchenne, or, perhaps, not seeing cases in which fatty changes are prominent (for they are rarely as marked as in our case), have not discoursed much upon the phenomena of lipomatosis. Duchenne says “the subcutaneous tissue contains very little fat.” Eulenberg, however, has brought these changes prominently forward, for in his monograph—founded upon his post-mortem researches—he says there is a great increase of subcutaneous fatty tissue, “most noticeable in places where nature has provided an abundant panniculus”; and, moreover, says of the intermuscular changes, that there is first a proliferation of connective tissue which is transformed into fatty tissue. I think that our case is a striking example of this pathology.

The bone lesions in this case are interesting in view of the tendency at present to connect bone and joint changes with diseases of the central nervous system. This speculation is not a new one, for it was a suggestion of Prof. J. K. Mitchell, years ago—so he is quoted,—that rheumatism may be of neurotic origin. Arthritis deformans has been recently attributed by Weber to a neurotic source, but the argument is largely from a therapeutic standpoint, viz.: as the disease does not improve on anti-rheumatic treatment, and does improve on general tonic and galvanic treatment, ergo—a neurotic origin. There are undoubted bone lesions, caused by disorders of the cerebro-spinal axis, as has been shown by Prof. H. C. Wood in a recent clinical lecture. He refers to bone changes in insanity, especially dementia paralytica, in hemiplegia, and locomotor ataxia. These changes are described as atrophy, hypertrophy, and inflammation. A true causative relation is shown, but we are wanting in an exact pathology as to what trophic centres, if any, are

affected, and in what manner. Bone lesions in pseudo-hypertrophic muscular paralysis have not been brought into any prominence in the investigations which I have consulted, and in many cases not even referred to. Scoliosis, which exists in one case, is mentioned by some and ascribed to the weakened state of the muscles—no doubt a true explanation, but such changes as I have described in the elbow and knee appear to be unique.

As a possible explanation of these impaired bones, I am reminded of the dictum of Duchenne. It was not the belief of this great clinician that pseudo-hypertrophic muscular paralysis was at all identical with progressive muscular atrophy, or that it was of central origin. It seems probable that the case before us may be an expression of that faulty development which overtakes, in some defective births, all tissues, and that pseudo-hypertrophic muscular paralysis, associated as it often is with impaired brain and nerve function, may be but a form, in some cases at least, of congenital deficiency of developmental power.

Dr. CHARLES K. MILLS said he had that day seen a case at the Philadelphia Polyclinic, interesting in connection with Dr. Lloyd's remarks on bone and joint lesion originating from the nervous system. The case was that of a mulatto woman who is changing to a bronze color, and in addition, the upper half of the body is affected with muscular atrophy. The joints, more particularly the smaller ones of the right hand, are swollen, rigid, and painful; the hands were in a half-closed position. She had perverted sensation in her face, loss of the sense of taste, difficulty of swallowing, and could only open her mouth half way on account of the muscles of deglutition and mastication being affected. The case illustrated the connection between muscular degeneration and joint and other trophic affections.

Dr. N. A. RANDOLPH read "A Note on the Behavior of Hydrobromic Acid and of Potassium Iodide in the Digestive Tract."

I venture to present before the Society a brief note upon a subject not strictly neurological, but having direct bearing upon the relations entertained to the processes of digestion by two drugs which are of interest to the neurologist.

In a series of artificial digestions, in which hydrobromic acid was present in the digestive mixture in amount corresponding to the therapeutic dose, I have noted:

(a) That salivary digestion was completely suspended, whereas

(b) The peptonization of proteid food-stuffs was in no wise retarded, the variation from the normal, if any, being toward an acceleration of this process.

It is evident, therefore, that, other things being equal, the appropriate time for the exhibition of this drug is immediately upon the cessation of salivary digestion within the stomach, or, in other words, upon the first formation of free acid within that viscus.

Recent studies¹ have shown that the acidity of the gastric contents, found even in quite early stages of digestion, is not due to the presence of *free* acid; and the ingenious observations of von den Velden² go far toward proving that the development of free acid within the stomach does not occur until from forty-five minutes to an hour after breakfast, and from one to two hours after dinner. These results were obtained chiefly by the use of methyanilin, violet and tropæolin bodies, delicately responsive, by color-change, to the presence of free acid. There is little doubt in my mind that hydrochloric acid is developed in the stomach at an earlier period than that above indicated, but it seems very probable that by immediate combination with albuminoid it loses somewhat its characteristic activity. This is illustrated by an observation made in the course of this study, namely, that the addition of small amounts of potassium iodide to dilute solutions of acid albumen containing two tenths of one per cent. of hydrochloric acid does not result in the liberation of iodine. The addition of the same amount of the iodide to the same quantity of an aqueous solution of hydrochloric acid of the same degree of activity results in an immediate liberation of iodine.

This interesting discovery of two stages of acidity in the gastric juice has, I believe, not yet been incorporated in the

¹ *Deutsch Archiv klin. Med.*, xxiii., 369. See also *Jahresb. u. d. Fortsch. d. Thier-Chemie*, 1880, p. 302, and Danilewsky, *Centralb. f. d. med. Wiss.*, 1880.

² *Zeitsch. f. physiol. Chemie*, iii., 205.

text-books. It serves to reconcile the contradictory opinions so frequently found as to the value to the economy of saliva as a digestive fluid, and explains the completely diverse results obtained by such careful workers as Frerichs¹ and Bidder and Schmidt.²

Besides an observance of the time-limitations just indicated, I would suggest the advisability of milk as a vehicle for the administration of hydrobromic acid. The curd thus formed is fine and flocculent, the mixture closely resembling buttermilk in taste and appearance, and in no wise suggesting medicine. I have taken as much as a tablespoonful of the dilute acid in a tumbler of milk without any repugnance. The milk used must be raw. With this, as with most other acids, boiled milk gives tough and bulky coagula.³

As regards the exhibition of the iodide of potassium, the rationale of its time-relations is altogether different. Although this drug is distinctly alkaline, its presence in a mixture of hydrated starch and saliva certainly does not suspend the action of the amylolytic ferment, nor, so far as I can determine, materially retard it.⁴ When, however, the iodide is added, even in very small amount, to a mixture of artificial gastric juice and egg albumen or fibrin, the rapidity of peptone formation, as determined by the nascent mercuric iodide reaction⁵ or by the biuret reaction and control test, is greatly diminished. This result is not due to a slight diminution in the acidity of the solution, caused by the addition of an alkaline body; for the same effect is noted when the acidity of the solution is at once again brought up to the normal degree. The pepsin is apparently but little, if at all, affected by the presence of the iodide, if we may judge by the indifference of ptyalin to the drug, and by the fact that quantities of the iodide corresponding to the maximal therapeutic dose neither entirely suspend

¹ Wagner, "Handwoerterbuch d. Physiologie," iii., a, 772.

² Bidder und Schmidt, "Verdadung u. Stoffwechsel," p. 27.

³ Randolph, "Verbal communication on differences between raw and boiled milk."—*Proc. Acad. Nat. Sci. of Phila.*, 1884.

⁴ Langley and Eves, *Four. of Physiologie*, iv., p. 19, have shown that although a distinctly alkaline medium retards salivary action, the presence of a proteid body in the digestive mixture will prevent this retardation.

⁵ Randolph, "A reaction common to peptone and bile salts."—*Proc. Acad. Nat. Sci. of Phila.*, 1884.

the peptic activity nor induce greater retardation of the digestion than do much smaller quantities. A slight effect is exerted by the iodide upon the proteid food-stuffs, evidenced in an increased toughness produced in, *e. g.*, fibrin, and, when the drug is abundantly present, in the acquisition by the albuminoid of a slight yellow tinge, due to staining by iodine, which is liberated by the free acid of the artificial gastric juice.

The most important factor in the delay of peptonization lies in the power possessed by potassium iodide, even in relatively minute quantity, of precipitating acid albumen in solutions which shall, after its addition, possess the normal degree of acidity of human or even canine gastric juice. The same may be said of potassium bromide and of several other analogous compounds. The precipitation effected by the iodide is so complete that, when solutions of acid albumen are thus treated and filtrated, the still acid filtrate yields no trace of proteid matter. This observation, which is doubtless old, though I have as yet been unable to find it recorded, tends to show that the time at which the administration of this drug is least liable to disturb digestion is either during or immediately after the ingestion of food.

There are several sources of error in attempts at deduction from the results of artificial digestion, as ordinarily performed. Thus the continued activity of a digestive fluid is largely conditioned by the removal of the products of its action soon after their formation. This occurs in the living viscus, but not in the test-tube of the experimenter. The maintenance of the normal temperature of the active stomach is, of course, readily accomplished, but the conscientious imitation of other factors in the normal digestive process implies not only a constant mechanical intermingling of food-stuff and digestive fluid, but the continued addition of small amounts of the digestive fluid itself. I have nearly perfected an apparatus which in a large degree obviates the difficulties just cited.

In artificial salivary digestions are conducted in a thin tube of fish-bladder, closed at one end, which is, by mechanical means, kept in gentle agitation. The contents of this tube are maintained at the proper temperature by a sur-

rounding body of warm water which is slowly but constantly changed. For gastric digestions the animal membrane is substituted by one offering equally great surface for dialysis, but resistant to peptic action. Despite, however, the inaccuracies attending existing methods of study, the following deductions from the facts, old and new, which are here presented, appear justifiable:

I. That the earliest production of free acid within the stomach is approximately three fourths of an hour after a meal; its appearance being still further delayed by the ingestion of food in large quantity;

II. That hydrobromic acid is liable to impede the digestion of starchy foods when administered within the interval just named; and

III. That iodide of potassium should be given at such time and in such dilution that its absorption shall be complete before the appearance of free acid within the gastric contents.

Dr. FRANCIS DERCUM said he thought the suggestion of using milk as a vehicle for hydrobromic acid a very good one.

Dr. S. WEIR MITCHELL spoke of the utility of hydrobromic acid given with bromide of potassium.

Dr. E. T. REICHERT referred to the paper recently written by Dr. H. C. Wood, on the "Use of Hydrobromic Acid in Epilepsy." He said it confirmed the conclusions which he had come to in a paper on the physiological action of hydrobromic acid, published some years ago. He believed that hydrobromic acid could be substituted for bromide of potassium, as it had the same physiological qualities as the bromide.

Dr. S. WEIR MITCHELL read a paper on "Œdema of Hysterical Hemiplegia and Unilateral Swelling in Hysteria Generally."

Dr. CHARLES K. MILLS said he had seen similar cases to those reported by Dr. Mitchell, but had not studied them closely. In a case of hysterio-epilepsy, with hemi-anæsthesia, etc., there was œdema of both limbs, but the swelling was most marked on the affected side, and was chiefly below the knee. In another case recently seen at the Philadelphia Hospital, the swelling of the limb was very decided.

In some cases of hysterical paraplegia he had noticed these swellings, but had not made them a special study. He also referred to a case of hysterical contracture of the wrist with marked œdema.

Dr. SINKLER spoke of having seen a number of cases of slight temporary swellings of the limbs in hysterical women. He mentioned a case of this kind which he had seen at the Orthopædic Hospital. It was that of a woman whose hands, feet, and legs would become, at times, slightly swollen, but it was temporary, as it would entirely disappear.

A stated meeting of the Society was held Monday evening, October 27, 1884, the Vice-President, Dr. CHARLES K. MILLS, in the chair.

The first paper was "On the Artificial Induction of Convulsive Seizures," by FRANCIS X. DERCUM, M.D., Ph.D., and ANDREW J. PARKER, M.D., Ph.D. (see page 579.) This paper was discussed by Drs. RANDOLPH, MASSEY, LLOYD, and MILLS.

The next paper was "On the Dietetic Factor in the Treatment of Angina Pectoris," by N. A. RANDOLPH, M.D.

There has recently been under my care a patient suffering from true angina, in whom, as is not seldom the case, any slight gastric irritation constituted the immediate exciting cause of the frequently recurrent paroxysms.

After the last attack there existed an inability to retain the lightest and simplest foods, their ingestion inducing not only nausea but much cardiac distress. Recourse was had, with advantage, to milk, partially digested by the commercial Extractum Pancreatis; but the flavor of the resultant preparation was unappetizing, and finally became repulsive to the patient, who whimsically described its taste as that of "stewed corpse."

To meet this emergency, there were devised two food products, which I have not seen described, and which, in practice, proved eminently satisfactory.

I. *Pancreatized Oysters*.—The oysters of an ordinary stew (containing milk) are removed and finely minced, then returned to the liquid portion of the stew. The whole is brought to a temperature of 100° F., the appropriate proportions of pancreatic extract and sodium bicarbonate are

added, and the mixture maintained at the temperature mentioned for thirty minutes, with occasional stirring. It is then strained and served, and forms not only a highly nutritious and palatable soup, but one which is retained by very irritable stomachs, and utilized with a minimum of digestive power. After boiling, to prevent the further action of the digestive ferment, gelatine may be added, and the mixture served cold, as a jelly. Cooked tomato, onion, celery, or other flavoring suited to the individual taste of the patient, may be added at the beginning of the artificial digestion, and the solid residue removed in the final process of straining, at which time it will be noticed that the mixed oysters originally added have been in great part dissolved.

II. *Pancreatized Milk-Toast*.—Ordinary milk-toast, in which there is an abundance of milk, when digested in the manner just described, becomes an almost homogeneous pulpy mass, which, when the crusts have been removed, is usually acceptably retained by the irritable stomach. In extreme cases, however, it may advantageously be strained and the fluid portion alone used, in which the partially peptonized solution of casein of the milk is reinforced by actually digested gluten and starch of the bread, together with a very little dextrin. Light plain sponge cake may be similarly digested, and occasionally forms a desirable change.

In conclusion I would express the hope that these rather homely suggestions may prove of value in other hands in extending the somewhat scanty bill of fare suited to patients suffering from gastric hyperæsthesia, and the various neurotic troubles of which such a condition may be the exciting cause.

Dr. DERCUM said he made an artificial preparation of beef, by using the pure gland of pepsin and beef, which was palatable, and answered the purpose admirably. He also used it by enemata.

Dr. G. BETTON MASSEY made a few remarks on "The Polarity of Currents of Medical Coils."

He called the attention of the Society to a singular omission made by the authors of most books on electro-therapeu-

tics. After stating in the introductions that induced currents were to-and-fro, with constantly changing polarity, they subsequently refer to these as having a fixed polarity, and make no explanation of this seeming impossibility. It is a fact readily tested by any one, that these coils furnish a current of one direction (the direct induced), at the opening of the battery circuit, and none at the closure. As it is difficult to close the battery circuit by hand in these batteries, without an irregular opening occurring at the same time, the currents due to this "raggedness" of the contact have been mistaken by some for inverse currents induced by the closure. Care, however, will eliminate this source of error, and prove that only currents produced in both primary and secondary coils at *opening* of the battery current are strong enough to be perceived by the senses. In the primary coil, whatever induction occurs at the closure, of course, goes through the cell as the short route. In the secondary coil this closure current is supposed to be inhibited by the simultaneously formed extra current of the primary coil. At any rate, it is not strong enough to be perceptible, and all, therefore, have genuine polarity, in our Faradic batteries.

Dr. LLOYD said that he did not think that Dr. Massey was quite just in his criticism on modern writers, as Erb and one or two others mentioned the facts in their works.

Dr. MASSEY said he was misunderstood, as he had said that *most* writers did not allude to it.

Dr. W. SINKLER said he wished to call the attention of the Society to a "Temperature Record of Each Side of the Body in a Case of Hemiplegia." He said, from the beginning to the end of the seizure the temperature on the affected side was from one fifth to one degree higher than on the other side.

The regular stated meeting was held November 24, 1884, the President, Dr. S. WEIR MITCHELL, in the chair.

Dr. Mitchell asked Dr. C. WOODNUT, resident physician in the Philadelphia Orthopædic Hospital and Infirmary for Nervous Diseases, to read the notes of a "Case of Erythremelalgia," which had been under Dr. Mitchell's care in the hospital.

J. C. R., æt. fifty-three married, blacksmith, family history good. Mother died at 72. Father still living. Six sisters still living and healthy; two died young. He has three children, all healthy. Has had scarlet-fever, and at twenty, variola, which left him slightly hard of hearing. Otherwise always perfectly well until September, 1881, when he first noticed pain on inner side of second toe of the left foot. One year after, the third toe was affected; later, the little, then the fourth, and about four months ago the great toe—all on the same side. They were all affected in the same manner—first a burning, then an aching pain, and some weeks after there would be discoloration of more or less of the toe. Great toe has been least affected of left foot, only feeling sore at tarso-metatarsal joint.

One year ago the pain began in the right foot, first appearing, as in the left foot, in the second toe. Now it affects second, third, and fourth toes, but not so badly as in the other foot.

Pain is not noticeably worse in wet or cold weather. It is worse at night when the feet get warm. The last seven months, however, this was not so marked. Has occasionally had twitchings, chiefly at night, in calf of left leg and bottom of left foot. About six years ago the arms began to ache at night, and do occasionally yet. Pain is confined, to muscles apparently, just above the elbows. Arms are not quite so strong as formerly, but attributes it to want of use. Nine years ago ran a harrow-tooth in outer side of bottom of left foot. It healed without difficulty, and left no apparent after-symptoms.

In January, 1883, had second toe of left foot amputated at proximal joint. It healed slowly, but relieved the pain not only in that toe, but also in the others.

The surgeon who removed the toe said that the amputated portion was "turned to gristle and had no circulation."

Has no history of syphilis nor alcoholism.

At present all the toes affected are discolored. The left foot, up to the ankle, is covered irregularly with reddish patches, sometimes dark and sometimes brighter, disappearing on pressure, and more or less sensitive. Little discoloration on right foot except at corners of nails, and the disease has not developed so rapidly in the right as in the left foot.

Foot more or less swollen to the ankle. "Tendons seem sore." Occasionally pain in outer left ankle. Occasionally whole leg aches, but greater pain is on the spots of redness.

Pain on walking in the ankle and in the bottom of foot, so can

only walk two or three blocks. Within the past few weeks a number of patches have appeared on the left leg, and one on the spine about the middle of dorsal region. For six or seven years has had occasional pains down the spine. It probably comes from a former sprain.

Also occasional sharp shooting pains at back of neck, on each side of spine, and down toward the shoulders, in the direction of brachial plexus. Not tender on pressure.

Sensation slightly diminished in the whole left limb, except on the patches of redness, where it is increased.

Elec. condition: no de. r.; response equal in both legs to galv. c.; Far. c., slightly diminished action in left leg below knee; sensation to both currents lessened in left limb. Measurements—r. calf, $13\frac{1}{8}$; l., $12\frac{1}{8}$; r. thigh, $20\frac{3}{4}$; l., $20\frac{1}{2}$.

Temp. r. $92\frac{3}{8}$, l. $93\frac{1}{8}$.

Dyn. r. 71, l. 70.

Appetite good, and, excepting pain, feels as well as ever. Urine, sp. g. 1.027; no albumen nor sugar.

His treatment has been absolute rest, massage, descending galvanic current, and hot and cold applications to the spine once daily. Chloride of gold and sodium, Fowler's solution, and ergot have also been used.

Nov. 21, 1884.—An irregular-shaped patch appeared on dorsum of each wrist, preceded for forty-eight hours by pain and soreness; disappeared after being out thirty-six hours.

Nov. 23d.—Redness appeared on each arm above elbow. Not preceded by pain, and appearing just after massage. Massage also brings it to view more plainly in the leg.

After the reading of the notes, Dr. MITCHELL brought the case before the Society.

Dr. CHARLES K. MILLS said he had seen quite a number of similar cases. His belief was that some at least of these cases were spinal in origin. The fact that some get well is not against this theory. A few years ago he had attended a lady who had all the symptoms of diffused myelitis, from unknown cause. He had also studied several cases of arsenical paralysis due to diffused myelitis. When the feet were pendent, in these cases, they would become hot, red in color, etc., like some of the cases of erythemelalgia. Dr. McBride of New York has described another type of cases, in which the limb is diminished in size, is cold, blue, etc.

Dr. MITCHELL said that he had no doubt but that some of the cases of erythemelalgia were spinal in origin. He was not so certain, though, as to the spinal condition. In some of the cases an hysterical condition is present.

Dr. GUY HINSDALE exhibited, with some remarks, a "Model of a Phantom Brain."

The model is of large proportions, and is intended to show the course of the fibres in the human brain and their relation to the cortex, to the ganglia, and to the spinal cord. The preparation has been recently purchased by Dr. S. Weir Mitchell, for the Mütter Museum of the College of Physicians of Philadelphia, and was constructed by Buechi, of Berne, Switzerland, under the supervision of Prof. Acby.

Its height is one hundred and twenty five centimetres; its width, seventy centimetres. The cortex is dotted over with numerous corks, two centimetres long, which are distributed in systematic order. The basal ganglia are seen in their appropriate places. The spinal cord, made up of ganglia and columns of nerves of different colors, is represented throughout a portion of the cervical region.

Dr. GUY HINSDALE read notes on "Potassium Bromate and its Action in Nine Cases of Epilepsy."

Bromate of potash, KBrO_3 , resembles in some respects chlorate of potash, while retaining the characteristics of bromides. It was used in the spring of 1881, by Dr. Weir Mitchell and Dr. Hinsdale. The latter made a personal test of the substance, and found that doses of ten, twenty, and thirty grains, three times a day, slowed the pulse decidedly, and depressed the heart, the larger doses causing purging and drowsiness. A single dose of forty grains caused watery discharges from the bowels and drowsiness.

In the nine cases of epilepsy its use was satisfactory in only one; doubtful in two; unsatisfactory in six. The drug is such an irritant poison, and depresses the heart to such a degree, that the substance had to be discontinued in most cases, although it evidently controlled the seizures.